## Application of ultrasound in pulseless electrical activity (PEA) cardiac arrest

Helaleh Rabiei<sup>1</sup>, Vafa Rahimi-Movaghar\*<sup>2</sup>

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While substantial efforts understanding of ventricular fibrillation (VF) pathophysiology have reduced deaths from ventricular fibrillation and ventricular tachycardia (1), pulseless electrical activity (PEA) during years have been associated with poor outcome. The focus of treatment in ventricular fibrillation is delivering shock; treatment of underlying etiology comes in the next stage, whereas in PEA, primary and timely diagnosis of the underlying acknowledged. cause is Meanwhile, CPR is ongoing. Therefore, studies and trials focusing on performing echocardiography alongside ACLS access this goal were conducted to assay the utility of echocardiography in cardiac arrest. Echocardiography could help the definite diagnosis of an underlying cause in some PEA cases and lead to appropriate intervention but it failed as a targeted intervention in a subset of patients and no study showed improvement in the outcome of these patients (2-5).

As an important result, these studies revealed that in a noticeable proportion of patients with the diagnosis of PEA or asystole, the echocardiography demonstrates cardiac motion (pseudo-PEA) and these patients have higher survival rates (4,6-8). Pseudo-PEA patients have a higher potential of ROSC compared to true PEA while therapeutic strategies in both cases are

similar. Some studies focused on the terminating prolonged resuscitation in true PEA subjects. Another approach is application of additional therapeutic strategies for pseudo-PEA patients when echocardiography does not determine specific causes of arrest and does not guide us to more effective key procedures.

PEA underlying causes are separated into primary and secondary forms. The secondary form includes the causes that result from an abrupt cessation of cardiac venous return, such as massive pulmonary embolism, acute malfunction of prosthetic valves, exsanguinations, and cardiac tamponade. Echocardiography during CPR is beneficial to detect secondary causes that include easily treatable, reversible pathologies associated with PEA (9). In primary PEA, none of those obvious mechanical factors is present, and ventricular muscle fails to produce an effective contraction despite continued electrical activity. The proximate mechanism for failure of electromechanical coupling is abnormal intracellular calcium metabolism, intracellular acidosis and adenosine triphosphate depletion that can occur because of acute myocardial ischemia, which is the major cause of cardiac arrest, toxins, and electrolyte imbalance. 70% of all cardiac arrests are caused by acute myocardial ischemia or massive pulmonary embolism (10). Diag-

<sup>&</sup>lt;sup>1</sup>. MD, Researcher, Sina Trauma and Surgery Research Center, Tehran University of Medical Sciences, Tehran, Iran. helale.rabiee@gmail.com <sup>2</sup>. (**Corresponding author**) MD, Professor of Neurosurgery, Research Vice Chancellor of Sina Trauma and Surgery Research Center, Department of Neurosurgery, Shariati Hospital, Tehran University of Medical Sciences, Tehran, Iran, & AOSpine research officer of Middle East, & Director of national spinal cord injury in Iran . v\_rahimi@sina.tums.ac.ir, v\_rahimi@yahoo.com

nosis of an acute ischemic stroke is possible by echocardiographic evaluation of ventricular wall function but in the setting of cardiac arrest, it becomes difficult and requires more studies. The review of the literature reveals that transthoracic echocardiography is highly specific but not highly sensitive to detect pulmonary embolism and few prospective studies have been conducted to evaluate the accuracy of the transthoracic echocardiography to detect pulmonary emboli in a cardiac arrest setting (11,12). In Breitkreutz et al. study, five pulmonary emboli out of one hundred arrests were diagnosed by echocardiography and in Hayhurst et al. study two of fifty arrests were suspected of pulmonary emboli. So, diagnosis and treatment of these two main etiologies should also be considered when there are no directly related echocardiographic findings, but other etiologies are ruled out. In a randomized, multicenter TROICA trial, all patients with asystole pulseless electrical activity and underwent immediately thrombolytic therapy with tenecteplase alone. It showed no significant improvement in survival (13) reports meta-analysis and case but suggested using thrombolysis combined with heparin as a successful therapy during prolonged pulmonary **CPR** in thromboembolic (PTE) patients (14,15). Perhaps applying a more selective strategy that specifies thrombolytic after using echocardiography and ruling out other reversible pathologies including pneumothorax, hypovolemia and tamponade improve survival.

Based on the pathophysiology for PEA arrest, the administration of calcium and adenosine might enhance cardiac contractility and increase the possibility of ROSC; as a result, physicians would have more time to carry out a precise work on the diagnosis of the underlying cause of arrest. There is a paucity of data on the use and possible appropriate timing of calcium administration in cardiac arrest in humans (16). By recognizing that the most common mechanism of PEA arrest in trauma is mostly due to anox-

ic cardiac arrest rather than coronary occlusion, calcium probably has a benefit, especially in PEA patients with ventricular contractions.

"Occult" ventricular fibrillation and difficulties with distinguishing between fine ventricular fibrillation and asystole may lead to delays of possibly lifesaving shocks (17). Pulseless electrical activity, occult VF and asystole are similar on EKG but require different treatments. The application of echo during CPR has been used as a diagnostic aid in this issue, too. Case reports indicated the benefit of ultrasound to detect occult VF that appeared asystole on EKG and allowed proper treatment with defibrillation (18,19). Now the questions are: Are there specific diagnostic ultrasound features to differentiate pseudo PEA vs. occult VF while both of them do not generate cardiac output? How could we be sure that patients diagnosed with pseudo-PEA are not occult VF? Do pseudo-PEA patients benefit from performing shock? In further researches, it is important to draw a sharp distinction between ultrasound findings of ineffective cardiac motion in PEA vs. cardiac fibrillation in occult VF. The American Heart Association's Guidelines do not recommend shocking in asystole or PEA but applying electrical defibrillation in PEA arrest patients in the compensatory stage, when an ultrasound shows cardiac motion, might be a point for further research.

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